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Understanding Scarring: Scarless Fetal Wound Healing as a Model

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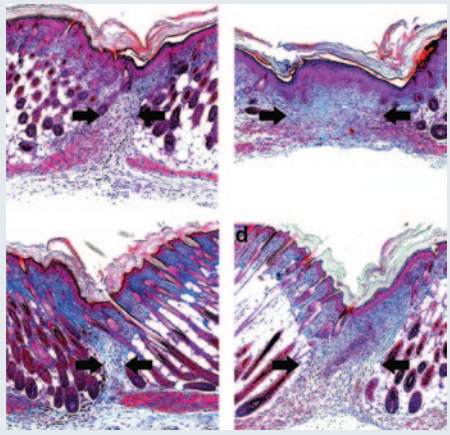


Scarring is a frequent unwanted consequence of full-thickness wound healing because such wounds heal via repair mechanisms as opposed to regenerative mechanisms. In certain situations, however, full-thickness wounds heal without scarring. Understanding the circumstance of scarless healing may lead to facilitation of the process. For example, *in utero*, early in fetal life, wounds heal by regeneration instead of repair (Larson *et al.*, 2010). This scarless fetal wound healing occurs across species, but it is age dependent—that is, at a certain time point in embryonic development, a switch is flipped that triggers scarring. In humans, this transition occurs in the last trimester of pregnancy (Wilgus, 2007).

Attempts have been made to understand the factors that early *in utero* allow for scarless healing. One important observation is that fetal wound healing occurs with minimal inflammation (Krummel *et al.*, 1987). By contrast, wound healing in late pregnancy and in adults is characterized by a robust inflammatory response and scar formation.

Using a well-characterized embryonic murine model in which scarless healing occurs at or before embryonic day 15 (E15) and scarring occurs at or after E18, Wulff *et al.* (2012, this issue) examined the potential role of mast cells in scar formation during fetal wound healing, based on evidence that mast cells are involved in fibrosis in conditions such as keloids and scleroderma. In evaluating cultured mast cells, normal skin, and wounded skin from E15 and E18 mice, the investigators found that mast cells displayed decreased numbers, maturity, and function (degranulation) in E15 skin, suggesting a role for mast cells in the promotion of scarring in E18 mice. Additional experiments investigated the results of injection of mast cell lysates from E15 and E18 mice and of mast cell–deficient mice. The resulting data further supported a role for mast cells in fibrosis in this model. The possible role of mast cells in the transition from scarless to fibrotic healing suggests a potential target in therapeutics.

Through the following questions, we examine this paper in greater detail. For brief answers, please refer to the supplementary information online <http://www.nature.com/jid/journal/v132/n2/suppinfo/jid2011444s1.html>.

REFERENCES

Krummel TM, Nelson JM, Diegelmann RF *et al.* (1987) Fetal response to injury in the rabbit. *J Pediatr Surg* 22:640–4

Larson BJ, Longaker MT, Lorenz HP (2010) Scarless fetal wound healing: a basic science review. *Plast Reconstr Surg* 126:1172–80

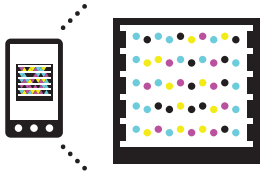
Wilgus TA (2007) Regenerative healing in fetal skin: a review of the literature. *Ostomy Wound Manage* 53:16–31

Wulff BC, Parent AE, Meleski MA *et al.* (2012) Mast cells contribute to scar formation during fetal wound healing. *J Invest Dermatol* 132:458–65

QUESTIONS

1. Describe scarring in fetal wound healing.
2. How do mast cells participate in wound healing?
3. How did the investigators carry out their study, and what were the results?
4. What were the conclusions and implications of the study?

For answers, visit our website or scan the adjacent tag.



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